Comparative study for three protocols of general anesthesia in bucks

Raffal A. Omar; Omar Khalid Aziz; M. J. Eesa and Bilal Shaker Abed
Department of Surgery and Obstetrics, College of Veterinary Medicine, Baghdad University, Iraq.
E-mail: raffal_omar@yahoo.com

Received: 5/5/2014
Accepted: 20/8/2017

Summary

The aim of this study was to evaluate three regimens in induction and maintenance of general anesthesia in local breed bucks and the investigation of the best protocol of them based on different parameters. Eighteen healthy adult bucks weighing (27.50 ± 0.682 kg), aged (1.989 ± 0.135 years) were used. Animals were divided randomly into three equal groups to undergo pre-experiment preparation. All groups were premedicated intravenously with (0.5 mg/ kg) Diazepam, ten min. Later, the induction for first group was done by Propofol (3 mg/kg B.W), second group was done by Ketamine Hcl (2 mg/kg B.W), while in the third group was done by Thiopental sodium (3mg/kg B.W). After intubation with suitable endotracheal tube, maintenance of the three groups was done by (1.3-1.5%) isoflurane and nitrous oxide with oxygen at ratio 2:1. All the three groups underwent the measurement of the following parameters; (Induction, recovery and duration times) Clinical parameters (heart rate, respiratory rate and body temperature); Clinical signs, analgesia, muscle relaxation; Liver enzyme (Aspartate Amino Transferase and Alanine Amino Transferase). The complete randomized design within statistical analysis system (SAS) program were used for statistical analysis as relevant with a significance level set at $P<0.05$. The results of this study showed that the induction of the anesthesia was smooth and uneventful in all the three groups and recovery time was shorter in 3rd group in comparison with 2nd and 1st groups. The three regimens showed no significant differences in heart rate. The 2nd group recorded a sharp decrease in respiratory rate and body temperature in comparison with 1st and 3rd groups. Analgesia was very suitable in all the three groups. There were significant changes among groups concerning Alanine Amino Transferase and, Aspartate Amino Transferase. It was concluded that all the three protocols were safe and caused no hazard effect on the animals.

Keywords: General anesthesia, Inhalation, Bucks, Propofol, Ketamine

---

Introduction

Anesthetics are among the most potent and rapidly acting drugs in common clinical use, and make one of the greatest advances of medicine allowing complicated surgeries to be performed safely (1). In veterinary practice, intravenous anaesthetic drugs were commonly used as induction agents to facilitate endotracheal intubation, whilst inhalation anaesthetic agents form the foundation for maintenance of general anaesthesia (2). Goats are gaining acceptance as an established model for biomedical research and for surgical training and teaching. They were used in medical, orthopedic, psychological, chemotherapeutic, and physiologic research (3). In addition to that, goats may be a better animal model for inhaled anesthetic pharmacokinetics in people (4).

Generally, goats can be particularly sensitive to stress and pain, therefore, it is important to perform procedures under adequate sedation with adequate analgesia (5). Sheep and goats were the poor relations where general anesthesia is concerned. However, equipment used for small animals is suitable for these species and, with attention to the special requirements of ruminants, sheep and goats can be anaesthetized successfully (6). The aim of this study was to evaluate three regimens in induction and maintenance of general anesthesia in bucks.

Materials and Methods

This study was carried out using eighteen healthy local breed bucks weighing (27. 50 ± 0.682 kg) and aged (1.989 ± 0.135 years). The animals were housed in the animal farm of the College of Veterinary Medicine, University of Baghdad, and maintained in individual kennels under normal environment including climate, management and feeding. The animals were dewormed by Ivermectin (overtone 1%) at a single dose (0.2 mg/kg B.W) administered

subcutaneously. Food and water were withheld (18 - 24) hrs; before each experiment, the animals were shifted to the hall were the experiment was carried out for acclimatization. Bucks were used in this experiment and divided randomly into three equal groups and were anesthetized as following: - in which all the groups was injected preanesthetics with Diazepam (0.5 mg/kg B.W) intravenously (7). In first group induction was done by Propofol (3mg/kg B.W) intravenously (8 and 9), second group induction was done by Ketamine Hcl (2mg/kg B.W) intravenously (10-12), while the induction agent of third group was done by Thiopental (3 mg/kg B.W) intravenously (13). After that maintenance was done by using Isoflurane at dose of 1.3% - 1.5% (10 and 14), and Nitrous Oxide: Oxygen at dose supplemented with 2:1 ratio of nitrous oxide to oxygen for all three groups anesthesia maintenance (15 and 16).

Physiological parameters included: - Rectal Temperature (°C); Respiratory and Heart rate which was recorded during and after anesthesia for the periods of 0, 10, 20, 30, 40, 50, and 60 minutes. The following parameters were measured during this experiment for all the groups: Induction time and its nature, Surgical anesthesia (Duration time) / minute, Recovery time/ minute; degree of analgesia, degree of muscle relaxation and Eye reflex.

Biochemical Parameters: Included determinations of Alanine Amino Transferase (ALT), and Aspartate Amino Transferase (AST) levels, in which blood samples were collected from jugular vein before, within and after anesthesia to determine AST and ALT levels. Statistical analysis was done by (SAS) Statistical Analysis System for determination the effect of different factors in the study of parameters. In this study, least significant difference (LSD) test was used to differentiate the significant differences between means (17).

Results and Discussion

Economic considerations and the limited number of anesthetics and analgesics licensed for use in small ruminants may dictate the use of a technique. Inhalational anesthesia is seldom feasible and economically justified, except when the economic value of the animal is high (18). However, some procedures, with economic justifications, were better performed under general anesthesia, and with certain precautions, general anesthesia can be carried out safely without complications (19).

According to (20) to minimize differences at induction the same premedication agent can be used at the same dosage since the type of drug used for premedication and the dosage used influences the amount of the induction agent required to achieve complete induction of general anesthesia. At induction period the results of this parameter show no significant differences $P<0.05$ between 1st group and 2nd group at induction time, (33.33±6.28; 23.33±2.42/ second) respectively (Table, 1). These results agree with (13) who found no significant differences between ketamine and propofol used for induction in cats. In contrast there was a significant differences $P<0.05$ between 1st group and 3rd group (33.33±6.28; 17.50±2.81/ second) (Table, 1). Our results disagree with (13) in which they found that Propofol is superior to thiopental and ketamine as an induction agent before halothane anaesthesia in goats. In addition, they mentioned that it provides uneventful recovery which is more rapid than thiopental or ketamine, so reduces anaesthetic risk and this might be due to the dose were used in our study different from (13) in which the increase in the dose always result in increasing the effect of thiopental sodium , this phenomena was coincided with (7).

Researchers (21) mentioned that γ-aminobutyric-acid (GABA) receptors in the central nervous system are thought to be a potential target site of action for a variety of general anesthetics. Extensive studies have shown that barbiturates, benzodiazepines, propofol, and volatile anesthetics such as halothane, isoflurane and sevoflurane, bind to their allosteric sites at GABA receptors, and potentiate GABA-activated chloride currents. These anesthetics are occasionally co-administered for induction or maintenance of anesthesia, presumably to facilitate a smooth and rapid induction or to reduce adverse effects by a single agent used at high doses.

Thiopental is an ultra-short acting barbiturate that provides smooth and rapid induction (22). While Propofol is a substituted alkyl phenol derivative that is believed to

facilitate gamma amino butyric acid activity in the brain. Its onset of action is almost instantaneous, because of high lipid solubility. Benzodiazepines and barbiturates were structurally distinct classes of drugs that were used clinically for their ability to depress CNS excitability in specific ways (23). While (24) believed that these drugs act at the postsynaptic GABA receptors of the CNS synapses. Benzodiazepines increase the frequency of chloride channel opening and barbiturates increase the duration of this open state.

The 2nd and 3rd groups reflected no significant differences between them. These results might be due to the effect of Diazepam premedication prior to induction with these agents because diazepam cause muscle relaxant and prevent the side effect of ketamine (muscle tone - spasm - contraction). These results did not agree with (13) who found that both Thiopental group and Propofol group showed a smooth uneventful induction of anesthesia in comparison with Ketamine group. Although 1st group recorded higher duration time (52.83±1.83/ min.), but it was non-significant with 3rd group (49.33±3.36/ min.), while 2nd group recorded a significant decrease in duration time group (49.33±3.36/ min.).

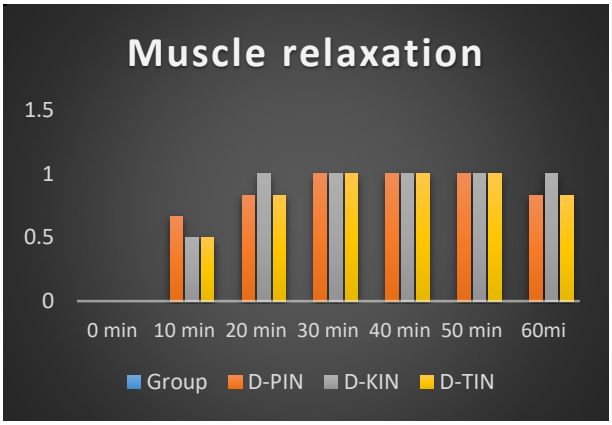
Although Propofol has a quick onset and smooth, rapid recovery might be due to rapid redistribution and rapid metabolism (25) but our results refer that in 1st group show significant increase in recovery time (15.33± 1.33/ second) and this could be because that Propofol is a complex drug with 3 half, or distribution of the drug from the blood to the tissues following administration is 2 to 3 minutes. The β half- life, or the elimination half - life, ranges from 30 to 60 minutes. The γ half - life, or terminal half - life during which the drug is eliminated from tissue fat, range from 300 to 700 minutes. Wake - up time following short - term administration is approximately 15 minutes (26) in comparison with 2nd and 3rd groups which recorded (10.67± 0.42 and 11.33± 1.56/ second) respectively. The later groups show no significant differences between them.

The results of muscle relaxation showed a good muscle relaxation start with the 1st group (66.7%) at 10 minutes in comparison with other two groups (50%), while 2nd group show a very good muscle relaxation at 20 minutes till 60 minutes (100%) in comparison with the other groups. Both 1st and 3rd groups show almost the same muscle relaxation degree all over the time of the experiment (Fig. 1). Generally, deep muscle relaxation was obvious in all the three groups from 20 minutes till 60 minutes (Table, 2).

Table, 1: shows the induction, recovery and duration times.

Treatment	Mean ± SE		
	Induction (time/sec.)	Duration time (time/min.)	Recovery period (time/min.)
1st group	33.33 ± 6.28 A	52.83 ± 1.83 A	15.33 ± 1.33 A
2nd group	23.33 ± 2.42 AB	40.67 ± 2.12 B	11.33 ± 1.56 B
3rd group	17.50 ± 2.81 B	49.33 ± 3.36 A	10.67 ± 0.42 B
LSD value	12.726 *	7.622 *	3.650 *

*(P<0.05). *Significant. Different capital letters revealed a significant difference at the level of (P<0.05) among groups.



Figure, 1: Shows muscle relaxation in the three groups.

Table, 2: shows evaluation of muscle relaxation.

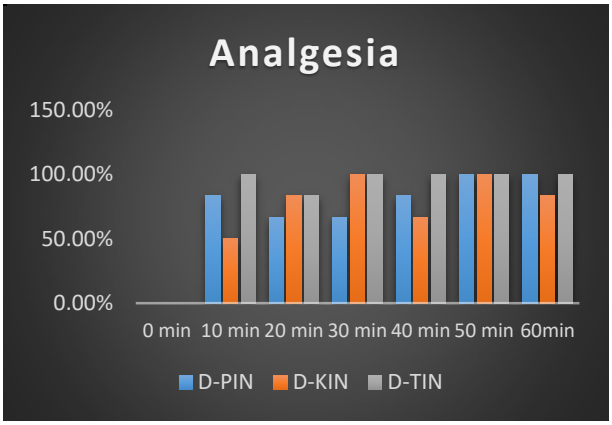
Time/ min. Groups	0	10	20	30	40	50	60
1st group	NR	MR	DR	DR	DR	DR	DR
2nd group	NR	MR	DR	DR	DR	DR	DR
3rd group	NR	MR	DR	DR	DR	DR	DR

NR: No Relaxation; LR: Light Relaxation; MR: Mild relaxation, and DR: Deep relaxation.

Although Ketamine was reported to be a poor muscle relaxant (27) usage of diazepam might be the cause of muscle relaxation observed, since diazepam is reported to be a good muscle relaxant (7), these results agreed with (2) who mention that the major drawback with Ketamine is inability to relax skeletal

muscles, which has given rise to a need to always co-administer it with benzodiazepines to facilitate adequate muscle relaxation to enable intubation and minimize muscle excitation (7 and 13), also our results agree with (28) who revealed to the use of diazepam with Ketamine aids muscle relaxation. According to (29 and 30) effect of muscle relaxation due to diazepam mediated through depression of polysynaptic musculoskeletal reflexes. Propofol usually possess anticonvulsant activity in addition to that it has anti-seizure properties (31 and 32) by inhibition calcium entry to muscle cells (33).

No pain had been shown among the three groups at zero time. 3rd group was the most affective; it reflected smooth analgesia (Fig .2), (Table, 3). Ketamine was considered to be a potent analgesic, blocking conduction of pain impulses to thalamic and cortical areas, (34) because of its action on the nucleus reticularis gigantocellularis in the brain stem, its modest affinity for opioid receptors and non-competitive N-Methyl D-Aspartate (NMDA) receptor antagonism (35). But Nitrous oxide which used in the present anesthetic technique may have enhanced NMDA receptor inhibition by ketamine because nitrous oxide too has been reported to exert NMDA antagonist properties (36).



Figure, 2: Shows Analgesia in the three groups.

Propofol has several mechanisms of action both through potentiating of GABA receptor activity, thereby slowing the channel closing time, and also acting as a sodium channel blocker usually used in clinical doses it is weak at controlling pain and have no analgesic effect, unless combined with analgesic drug (37). Usually ketamine combined with drugs such as xylazine and/or diazepam to improve analgesia and muscle relaxation of anesthetic regimes (38).

Table, 3: Shows Evaluation of analgesia in the three groups.

Time/ min. Groups	0	10	20	30	40	50	60
1st group	NA	DA	MA	MA	DA	DA	DA
2nd group	NA	MA	DA	DA	MA	DA	DA
3rd group	NA	DA	DA	DA	DA	DA	DA

NA: No Analgesia; LA: Light Analgesia; Ma: Mild Analgesia, and DA: Deep Analgesia.

The occurrence of mild analgesia could be explained as the mechanism of volatile anesthetic enhancement of pain sensitivity as being not known. According to one hypothesis, isoflurane inhibits nicotinic receptors that are a presynaptic 'gain control' mechanism tonically regulating norepinephrine release in the spinal cord. There is extensive evidence for nicotinic facilitation of norepinephrine release in the spinal cord. It is further thought that the norepinephrine released has analgesic action through activation of α2-adrenergic receptors. In support of this hypothesis, both depletion of norepinephrine with neurotoxin and intrathecal injection of the α2-adrenergic antagonist, yohimbine, prevent nicotinic antinociception. These findings taken together raise the possibility that isoflurane enhances pain sensitivity by inhibiting nicotinic acetylcholine receptors in the spinal cord, with the net effect of reducing norepinephrine release (39). In another word enhanced release of norepinephrine in the spinal cord is thought to be one mechanism by which nicotine induces analgesia. In all the three groups, observation of the eye reflexes (palpebral and corneal reflexes) were abolished completely after 20 minutes from injection (Table, 4).

Table, 4: shows Eye reflex in all the three groups.

Time / min. Group	0	10	20	30	40	50	60
1st group	-	-	+	+	+	+	+
2nd group	-	-	+	+	+	+	+
3rd group	-	-	+	+	+	+	+

(-)ve: presence of the reflex, (+)ve: abolish.

Ketamine is a dissociative anesthesia characterized by a cataleptic state in which, the eyes remain open with slow nystagmicgaze.

Therefore all reflexes remain active, such as corneal, which is more difficult to assess the anesthetic plane, the abolishment of eye reflexes attributed to the effect of thiopental on the CNS by effect on the level of thalamus where they inhibit ascending conduction it the reticular formation impulses to the cortex analgesia and reflex response to stimuli is not abolished until an appreciably greater depth of unconsciousness is reached than is required with many other agents (27).

It is well known that the common adverse effects associated with isoflurane include respiratory depression, hypotension and reduced cardiac output (40), but our results did not agree with (41) who found that inhalation anesthetic agent expected to remain the same within any species if physiological states like body temperature, blood pressure, haematocrit and tissue perfusion were maintained within normal levels.

Respiratory rates in all groups were not affected severely in which there was no apnoea in all the three groups. In comparing the propofol with thiopental no significant differences were noticed during all periods. According to (25) the degree of respiratory depression with propofol is similar to that of thiopentone. At 20 and 50 minutes, there was significant variation in respiratory rate between 2nd group and other groups. Within 2nd group there was significant differences at zero, 10 and 50 minutes, it recorded (14.83 0.65 breath/ minutes), at the later time (23.50±0.56 and 24.17±031/minute), and significant with 1st group (23.50±0.56 and 22.00±0.73/ minute). At 10, 30 and 60 minute there was no significant difference among the three groups. At 20 and 40 minute, there was a significant decrease in respiratory rate in 2nd group (15.50±0.72 and 15.50±1.08/ minute), in comparison with other two groups (Table, 5).

The depression in respiration is a consequence of depressing of brainstem and it could be postulated as the greater the depression of the brain (42 and 43). There were no significant differences within 1st group at period lasted from zero time to 60 minutes. The 2nd group showed a significant different decrease in respiratory rate at 50 minute in comparison with zero time and 10 minutes. However, these records were not significant in comparison with 20, 30, 40 and 60 minute. The 3rd group revealed a significant decrease in respiratory rate at period lasted from 10 to 60 minute in comparison with zero time and this result agreed with most of research which reported that thiopentone sodium had a depressing effect on respiratory rate (44) because of its direct depressing effect on the respiratory center, our results does not agree with (45) in which the decreasing of respiratory rate continued till 30 minutes.

Table, 5: Effect of the three protocols on the respiratory rate.

Time Min.	Mean ± SE of Respiratory rate			LSD value
	1st group	2nd group	3rd group	
0	22.00 ±0.73 Ba	23.50 ±0.56 ABa	24.17 ±0.31 Aa	1.691*
10	20.00 ±1.46 Aa	17.67 ±1.05 Ab	20.83 ± 0.91 A	3.511 NS
20	19.67 ±1.20 Aa	15.50 ±0.72 Bbc	19.33 ±0.42 Ab	2.545*
30	19.00 ±0.85 Aa	16.83 ±1.11 Abc	19.33 ±1.12 Ab	3.115 NS
40	18.67 ±0.98 Aa	15.50 ±1.08 Bbc	19.33 ±0.84 Ab	2.949*
50	19.00 ±1.12 Aa	14.83 ±0.65 Bc	20.00 ±0.89 Ab	2.748*
60	19.67 ±1.58 Aa	16.67 ±1.23 Abc	19.33 ±0.98 Ab	3.891 NS
LSD value	3.362 NS	2.722 *	2.385 *	----

*(P<0.05). NS=No significance. * Significant. Different capital letters revealed a significant difference at the level of (P<0.05) among groups. Different small letters revealed a significant difference at the level of (P<0.05) within group.

There were no significant differences in heart rate among the three groups at all periods, but there were significant differences within animals related to 1st and 3rd groups. Both drugs midazolam and diazepam caused mild decrease in heart rate (7 and 46). Ketamine was able to temporary counteract the bradycardia produced by midazolam and diazepam, where ketamine stimulates the central sympathetic outflow, which in turn, causes stimulated of the heart (47), (Table, 6).

Biochemical examination for ALT and AST levels revealed a significant increase in ALT level in 2nd group among groups during the period lasted from zero the time (20.70±2.22 IU/L), till 60 minutes (23.46± 1.55 IU/L) (Table, 7), while there were no significant

differences within propofol, ketamine and thiopental group from the period lasted from zero to 60 minutes.

Table, 6: Effect of the three protocols on heart rate.

Time Min.	Mean ± SE of Heart rate			LSD value
	1st group	2nd group	3rd group	
0	79.00±0.68 Aa	81.83±0.94 Aa	81.17±1.17 A a	2.871 NS
10	72.00±1.86 Ab	70.67±3.17 Ab	70.33±2.99 Ab	8.265 NS
20	70.67±1.61 Ab	74.00±3.76 Aab	70.50±2.55 Ab	8.388 NS
30	69.67±1.30 Ab	67.67±1.67 Ab	69.00±1.61 Ab	4.633 NS
40	78.17±2.42 Ab	72.33 ±4.16 Ab	73.17±2.92 Ab	9.666 NS
50	71.00±2.29 Ab	66.67±2.51 Ab	67.67±3.07 Ab	7.979 NS
60	71.33±1.83 Ab	69.83±2.99 Ab	70.83±3.29 Ab	8.376 NS
LSD value	5.064 *	8.449 *	7.540 *	----

*(P<0.05). NS=No significance. *Significant. Different capital letters revealed a significant difference at the level of (P<0.05) among groups. Different small letters revealed a significant difference at the level of (P<0.05) within group.

Table, 7: Show effect the treatment on the level of ALT.

Time Min.	Mean ± SE of ALT			LSD value
	1st group	2nd group	3rd group	
0	15.02±1.36 B a	20.70±2.22 A a	12.70±0.82 B a	4.759 *
10	15.73±1.27 B a	23.33±2.26 A a	12.10±1.02 B a	4.848 *
20	15.28±0.81 B a	21.58±2.15 A a	12.26±0.92 B a	4.319 *
30	13.86±0.73 B a	24.15±2.27 A a	11.78±0.57 B a	4.278 *
40	15.30±1.32 B a	22.68±2.24 A a	12.05±0.79 B a	4.747 *
50	14.25±1.03 B a	23.67±1.69 A a	12.60±1.06 B a	3.925 *
60	16.35±1.08 A	23.46±1.55 A a	14.35±1.12 A a	4.359 *
LSD value	3.195 NS	5.966 NS	4.882 NS	----

*(P<0.05), NS=No significance. *Significant. Different capital letters revealed a significant difference at the level of (P<0.05) among groups. Different small letters revealed a significant difference at the level of (P<0.05) within group.

3rd group recorded a significant decrease in AST Level (Table, 8) from zero time (71.81 ± 5.12) to 60 minutes (67.05±6.86 IU/L). While both propofol and ketamine groups showed no significant differences between them. Although there was no significant difference between ketamine and thiopental at period lasted from zero to 40 minute, there were no significant differences within propofol, ketamine and thiopental groups from the period lasted from zero to 60 minutes. Our results agree with (48) who studied the effect of halothane and isoflurane anesthesia in goats and found that Serum Sorbitol Dehydrogenase (SDH), Gamma-Glutamyl Transferase (GGT), and Alkaline Phosphatase (ALP) activities and Billirubin concentration did not increase after anesthesia and their results suggested that use of halothane or isoflurane for anesthesia in young healthy goats was unlikely to cause hepatic injury.

Table, 8: Show effect the treatment on the level of AST.

Time Min.	Mean ± SE of AST			LSD value
	1st group	2nd group	3rd group	
0	168.23±31.73 A a	107.10±24.92 AB a	71.81 ±5.12 B a	70.786 *
10	133.07±19.71 A a	111.67±15.62 AB a	69.30±5.05 B a	44.672 *
20	144.06±16.37 A a	107.83±17.36 AB a	67.08 ±5.71 B a	42.715 *
30	137.41±15.50 A a	112.03±20.76 AB a	70.13±5.96 B a	46.280 *
40	138.58±13.67 A a	111.36±17.68 A a	70.18 ±5.14 B a	39.917 *
50	143.25±17.52 A a	115.36±21.04 AB a	67.01±7.16 B a	49.273 *
60	127.48±15.31 A a	108.20±15.73 A a	67.05 ±6.86 B a	40.023 *
LSD value	55.678 NS	55.350 NS	16.985 NS	----

*(P<0.05), NS=No significance. *Significant. Different capital letters revealed a significant difference at the level of (P<0.05) among groups. Different small letters revealed a significant difference at the level of (P<0.05) within group.

References

1. Erasso, D.M. (2011). Differential Effects of Isoflurane and Propofol Anesthesia on Neurogenesis in Young and Aged Rats. PhD thesis. College of Medicine - University of South Florida.
2. Dzikiti, T.B. (2013). Intravenous anaesthesia in goats: A review. J. S. Afr. Vet. Assoc., 84(1):1-8.
3. Fulton, L.K.; Clarke, M.S. and Jr., H.F. (1994). The Goat as a Model for Biomedical Research and Teaching. ILAR. J., 36(2):21-29.

4. Soares, J.H.N.; Fukushima, F.B.; Hodges, F.B.J. and Liu, H. (2012). Solubility of Haloether Anesthetics in Human and Animal Blood. Anesthesiol., 117(1):48-55.
5. Caulkett, N. (2003). Anesthesia of ruminants. Veterinary rounds. 3(2):31-36.
6. Taylor, P.M. (1991). Anaesthesia in sheep and goats, In Practice. 13:31-36.
7. Ghurashi, M.A.H.; Seri, H.I.; Bakheit, A.H.; Ashwag, E.A.M. and Abakar, J.A. (2009). Evaluation of Ketamine/ diazepam Anaesthesia for Performing Surgery in Desert Goats under Field Condition Australian J. Basic and App. Sci., 3(2):455-459.
8. Hellebrekers, L.J.; de Boer, E.J.; Van Zuylen, M.A. and Vosmeer, H. (1997). A comparison between medetomidine-ketamine and medetomidine-propofol anesthesia in rabbits. Lab. Anim., (Lond.) 31:58–69.
9. Bertens, A.P.; Booiij, L.H.; Flecknell, P.A. and Lagerweij, E. (1993). Anaesthesia, analgesia and euthanasia. In van Zutphen L F M, Baumans V, Beynen A C (eds) Principles of laboratory animal science (1st ed). Elsevier, Amsterdam. Pp: 267–298.
10. Caulkett, N. (2003). Anaesthesia of ruminants, Large Animal Veterinary Rounds 3(2), viewed 20 June 2012, from http://www.larounds.ca/crus/laveng0203.pdf.
11. Taylor, P.M. (1998). Anesthesia in sheep and goats. In Practice. January, Pp:31-36.
12. Ewing, K.K. (1990). Anesthesia techniques in sheep and goats. Veterinary Clinics of North America: M. Smith, ed. Philadelphia: W. B. Saunders. Food Anim. Prac., 6(3):759-778.
13. Prassinos, N.N.; Galatos, A.D. and Raptopoulos, D. (2005). A comparison of propofol, thiopental or ketamine as induction agents in goats. Vet. Anaesth. Analg., 32: 289–296.
14. Papich, M.G. (2011). Saunders Handbook of Veterinary Drugs (small and large animals), 3rd ed. Elsevier; Saunders.USA. Pp:825.
15. Hayama, S.; Terazawa, F. and Suzuk, M. (2006). Immobilization with a single dose of ketamine hydrochloride and a combination of xylazine hydrochloride ketamine hydrochloride and antagonism by yohimbine hydrochloride in the Japanese monkey (*Macaca fuscata*). Primates, 30(1):75–79.
16. Flecknell, P. (2005). Clinical experience with NSAIDs in Macaques. Lab. Primate Newsl. 44(1):4.
17. SAS. (2010). Statistical Analysis System, User's Guide. Statistical. Version 9.1th ed. SAS. Inst. Inc. Cary. N.C. USA.
18. Galatos, A.D. (2011). Anesthesia and analgesia in sheep and goats, Veterinary Clinics of North America: Food Anim., 27:47–59.
19. Lee, T.L.; Ang, S.B.; Dambisya, Y.M.; Adaikan, G.P. and Lau, L.C. (1999). The effect of propofol on human gastric and colonic muscle contractions. Anesth. Analg. 89:1246–1249.
20. Dzikitia, T.B.; Chanaiwab, S.; Mpondab, P.; Sigaukeb, C. and Dzikitic, L.N. (2007). Comparison of quality of induction of anaesthesia between intramuscularly administered ketamine, intravenously administered ketamine and intravenously administered propofol in xylazine premedicated cats. J. S. Afr. Vet. Ass., 78(4): 201-204.
21. Luo A. and Sugiyama, K. (2000). Propofol combined with diazepam synergistically potentiates the GABA-activated chloride current in rat sensory neurons. Chin. Med. J. (Eng.). 113(9):840-843.
22. Valverde, A. and Doherty, T.J. (2008). Anesthesia and analgesia in ruminants, in R. Fish, P.J. Danneman, M. Brown and A. Karas (eds.), Anesthesia and analgesia in laboratory animals. Academic Press, 2nded., Pp:385-411.
23. Study, R.E. and Barker, J.L. (1981). Diazepam and (-)-pentobarbital: Fluctuation analysis reveals different mechanisms for potentiation of y-aminobutyric acid responses in cultured central neurons (benzodiazepines/ barbiturates). Proc. Natl. Acad. Sci. USA. 78(11):7180-7184.
24. Koshy, T.A.; Mahabala, T.H.; Srikantu, J. and Sanmathi, S. (2003). Thiopentone – midazolam mixture as an induction agent for general anasthesia on 'in-patients'. Indian J. Anaesth. 47(2):129-133.
25. Flaherty, D. (2009). Anaesthetic Drugs – chapter 6. In Anaesthesia for Veterinary Nurses. 1st edition, Edited by: Welsh, L.; Wiley – Blackwell, A John Wiley & Sons, Ltd., pp 121 – 161.

26. Brusco, L. (2002). Choice of sedation for critically ill patients: A partial approach. Advanced studies in medicine 2(9):343-350.

27. Hall, L.W.; Clarke, K.W. and Trim, C.M. (2001). Veterinary anaesthesia 10th ed., W.B Saunders Co., London. Pp:84-87, 352-353.

28. Ismail, Z.H.; Jawasreh, K, and Al-Majali, A. (2010). Effect of xylazine–ketamine–diazepam anesthesia on certain clinical and arterial blood gas parameters in sheep and goats. Comp. Clin. Pathol ., 19:11–14.

29. Lakoski J.M.; Murray W.B. and Kenny J.M. (2000). The Advantages and Limitations of Calmatives for Use as a Non-Lethal Technique. Applied Research Laboratory, www.mindfully.org.

30. Cantwell, S.L. (2001). Ferret, rabbit, and rodent anesthesia. Vete. Clin. North Am. Exotic Anim. Pract., 4:169- 191.

31. Kotani, Y.; Shimazawa, M.; Yoshimura, S.; Iwama, T. and Hara, H. (2008). The Experimental and Clinical Pharmacology of Propofol, an Anesthetic Agent with Neuroprotective Properties. CNS Neurosci. and Therap.14(2):95-106.

32. Volti, G.L.; Murabito P.; Attaguile G.; Rodella L.F.; Astuto M.; Giacomo C.D. and Gullo A. (2006). Antioxidant properties of propofol: when oxidative stress sleeps with patients. 5:25-32

33. Horibe M.; Kondo I.; Damron D.S. and Murray P.A. (2001). Propofol attenuates capacitative calcium entry in pulmonary artery smooth muscle cells. Anesthesiol., 95:681-688.

34. Meyer, D.J. and Harvey J.W. (1998). Veterinary Laboratory Medicine. Interpretation and Diagnosis. 2nd ed. W.B. Saunders Company. Philadelphia, London, Toronto, Montreal, Sydney, Tokyo, Pp:157-187.

35. Bhattacharya, D. and Banerjee, A. (2004). A comparative study of clinical effects of Intrathecal hyperbaric bupivacaine and Ketamine in hyperbaric solution. Indian J. Anaesth., 48(2):116-120.

36. Jevtović-Todorović, V.; Todorović, S.M.; Mennerick, S.; Powell, S.; Dikranian, K.; Benshoff, N.; Zorumski, C.F. and Olney, J.W. (1998). Nitrous oxide (laughing gas) is an NMDA antagonist, neuroprotectant and neurotoxin. Nat. Med., 4(4):460-463.

37. Kanazawa, M.; Nitta M.; Murata T. and Suzuki T. (2006). Increased dosage of propofol in anesthesia induction cannot control the patient's responses to Insertion of a laryngeal mask airway. Tokai J. Exp. Clin. Med., 31:35-38.

38. Mohammed, M.S. (2011). A comparison of four injectable anesthetic regimes in detomidine premedicated donkeys. M.Sc., College of Veterinary Medicine - University of Baghdad, Veterinary surgery (Veterinary anesthesia).

39. Flood, P. and Rowley, T.J. (2008). Isoflurane Prevents Nicotine-Evoked Norepinephrine Release from the Mouse Spinal Cord at Low Clinical Concentrations. Int. Anesth. Res. Society. 107(3):885-889.

40. Hikasa, Y.; Hokushini, S.; Takase, K. and Ogasawara, S. (2002). Cardiopulmonary, haematological, serum biochemical and behavioral effects of sevoflurane compared with isofurane or halothane in spontaneously ventilating goats, Small Rum. Res., 43:167-178.

41. Quasha, A.L.; Eger, E.I. and Tinker J.H. (1980). Determination and applications of MAC. Anesthesiology. 53(4):315-334.

42. Kul, M.; Koc, Y.; Alkan, F. and Ogurtan, Z. (2000). The effects of xylazine-ketamine and diazepam-ketamine on arterial blood pressure and blood gases in dogs. J. Vet. Res., 4:124-132.

43. Upton, R.N.; Martinez, A.M. and Grant, C. (2009). Comparison of the sedation properties of CNS 7056, Midazolam, and propofol in sheep. British J. Anaesth. 103(6):848-57.

44. Gaudy. J.H.; Dauthier, C.; Galliot, M.; Ferracci, F. and Boitier, J.F. (1983). Ventilatory effects produced by the i.v. administration of incremental doses of thiopentone in the dog. British J. Anaesth., 55:977-984.

45. Ghurashi, M.A.H.; Sobair, A.T.H.; Seri, H.I. and Ashwag, E.A.M. (2008). Some anaesthetic and physiological effects of Thiopentone sodium in goat kids. Assiut Vet. Med. J., 53(115):169-178.

46. Muir, W.W. and Mason, D.E. (1993). Effect of diazepam, acepromazine, detomidine and Xylazine on Thiamylal anaesthesia in horses. J. Am. Vet. Med. Assoc., 203(7):1031-1038.

47. Richard, D.H.; Mary, J.M. and Richard, A.H. (2009). Lippincott's Illustrated Reviews: Pharmacology. 4th ed. Lippincott Williams and Wilkins. Pp:137-138.

48. McEwen, M.M.; Gleed, R.D.; Ludders, J.W.; Stoko, T.; Del Piero, F. and Erb, H.N. (2000). Hepatic effects of halothane and isoflurane anesthesia in goats. J. Am. Vet. Med. Assoc., 217(11):1697-700.

دراسة مقارنة لثلاث بروتوكولات للتخدير العام في الماعز

رفل عبد الدائم عمر و عمر خالد عزيز و محمد جواد عيسى و بلال شاكر عبد

فرع الجراحة والتوليد، كلية الطب البيطري، جامعة بغداد، العراق.

E-mail:raffal_omar@yahoo.com

الخلاصة

هدفت الدراسة إلى تقييم ثلاثة برامج تخدير عامة تخص الإحداث والإدامة في الماعز المحلي والتحقيق لإيجاد أفضل بروتوكول منها بالاعتماد على عدة معايير. ثمانية عشر من ذكر الماعز محلي بوزن (27.50 ± 0.682) كغم وبعمر (1.989 ± 0.135) سنة تم استخدامها في هذه التجربة. قسمت الماعز عشوائيا إلى ثلاث مجاميع متساوية ضمت كل مجموعة ست حيوانات خضعت جميعها للتحضيرات المسبقة ما قبل إجراء التجربة. حقنت جميع حيوانات المجاميع الثلاثة وريديا بالدايزيبام بجرعة 0.5 ملغم/ كغم كعلاج تمهيدي بعد مرور عشرة دقائق تم الإحداث عن طريق الحقن الوريدي في المجموعة الأولى باستخدام البروبوفول وبجرعة 3 ملغ/ كغم وفي المجموعة الثانية الكيتامين وبجرعة 2 ملغم/ كغم، أما المجموعة الثالثة فقد تم بحقن الثايوبنتال صوديوم وريدياً بجرعة 3 ملغم/ كغم، وبعد أن أدخل أنبوب الرغامي تمت إدامة التخدير في المجاميع الثلاث عن طريق التخدير الاستنشاقي باستخدام الايزوفلورين (1.3-1.5)% وأوكسيد النايتروجين والأوكسجين بنسبة 1:2. اعتمدت المعايير الاتية في تقييم كفاءة المجاميع الثلاث وكانت كالاتي (الإحداث، مدة الافاقة، وقت التخدير الجراحي)، المعايير السريرية شملت عدد ضربات القلب/ دقيقة، سرعة التنفس/ دقيقة ودرجة حرارة الجسم/ درجة مئوية، العلامات السريرية، درجة تسكين الألم، درجة ارتخاء العضلات، مستوى انزيمات الكبد (انزيم الالنين الناقل وانزيم الاسبارتيت الناقل). استخدم تصميم التجربة العشوائي الكامل ضمن برنامج التحليل الاحصائي SAS لغرض تحليل نتائج التجربة احصائياً عند مستوى معنوي $P < 0.05$. أظهرت نتائج هذه الدراسة أن الإحداث كان سلساً وهادئاً في المجاميع الثلاث وكان وقت الافاقة أقصر في المجموعة الثالثة مقارنة بالمجموعة الثانية والأولى. لم تظهر المجاميع الثلاث اية فروقات مهمة احصائيا فيما يتعلق بمعدل ضربات القلب في حين سجلت المجموعة الثانية هبوطا حادا في معدلات سرعة التنفس ودرجة الحرارة مقارنة بالمجموعة الأولى والثالثة. كانت درجة التسكين للألم مناسبة في المجاميع الثلاث. ظهرت تغييرات مهمة احصائياً بين المجاميع الثلاث فيما يتعلق بمستوى انزيمات الكبد. ممكن أن نستنتج من هذه الدراسة ان البروتوكول في المجاميع الثلاث كان آمناً ولم يسبب أي تأثير مؤذٍ على الحيوانات

الكلمات المفتاحية: التخدير العام، استنشاقي ، ذكر الماعز، البروبوفول، الكيتامين.